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Mathematical Analysis of Efficacy of Condom as a Contraceptive on the Transmission of Chlamydia Disease

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Abstract: Chlamydia disease caused by the Chlamydia trachomatis is one of the major sexually transmitted infectious diseases globally. The progression of this disease has deadly effects cumulating into millions of death. Chlamydia causes numerous complications such as infertility in female, chronic pelvic pain and inflammation. Previous studies did not consider the effects of undetected infected individuals on the dynamic spread of the disease. Hence, this work investigated the effects of undetected infected individuals and efficacy of condom as a contraceptive in the dynamic spread of the disease. A six compartmental model to study the dynamic spread of chlamydia disease was developed using system of ordinary differential equation. The population was divided into susceptible, exposed, infected undetected symptomatic, infected detected symptomatic, infected asymptomatic and recovered individuals. The well-posedness of the model was investigated by the positivity of solution technique. Basic reproduction number (R_0) was computed using Next Generation Matrix Method. The endemic equilibrium was investigated by the use of Lyapunov function. The results showed that the disease free equilibrium was stable whenever the basic reproduction number is less than unity (R_0<1). The endemic equilibrium was found to be stable as a results of constructed Lyapunov function being negative definite. Numerical analysis showed that increasing in the undetected infected individuals enhanced the spread of the disease. Findings showed that undetected infected individuals played a vital role in the spread of Chlamydia disease. Moreover, using condom as a contraceptive reduced the spread of the disease.

Keywords: Chlamydia disease, model formulation, disease free equilibrium and numerical simulations

1. Introduction

Chlamydia infection is the most common sexually transmitted disease (STD) in human worldwide especially in European countries and the united states of American which is caused by the bacterium Chlamydia trachomaties. About 92 million Chlamydia infectious diseases occurred worldwide in 1990 include 50 million women and 42 million men. Any sexually active person can be infected with Chlamydia risk of infection varies considerably depending on the ages. It was observed that people in the age group 20-24 years are at high risk of being infected, and tends to be the most sexually active in a population with 36% of this age group by Sharma and [9]. [8] presented spatial temporal mathematical model of Chlamydia infection host immune response and movement of infectious particles. Numerical solution and model analysis are carried out and he presents a hypothesis regarding the potential for treatment and prevention of infection by increasing Chlamydia particle motility. [11, 13] studied screening for Chlamydia, his aimed at the reduction of these infection and subsequent complication selective screening may increase the cost effectiveness of a screening progamme. Few population based systematic screening programme have been carried out and attempt to validate selective screening criteria have shown poor performance. His study describes the development of a population rate for estimating the risk of Chlamydia infection as a basis for selective screening. [10, 14, 16, 7] used new approach to mathematical modeling that tests intervention efforts on Chlamydia. They aim was to produce a simple model that can be used when new data comes to hand without the need to re-run the stimulation. A simple model was developed to study the effect of intervention in lowering rates of chlamydia in a high-risk population of 16 to 24 years olds. Parameters are informed by the best available data. The model was verified by raining it backward in time to see if it correctly retrofits rates of chlamydia in the past. The model predicted that chlamydia would disappear long term if there were 45% condom use annual check-ups and 23.5% successful contact tracing among the high-risk 16–24 year old age group. The model expression can be applied readily to different population of interest and to address specific question, indicating that model is a quick and easy tool to apply in public health policy making.

[1, 3, 8, 6, 9] developed epidemiological model in the human population with Chlamydia trachomatis. Their model incorporated the vaccination class and investigated the role played by some control strategies in the dynamics of the disease and the optimal control of the model shows the effect of different strategies in the transmission dynamics of the disease and the cost effectiveness of each control pair. It was observed that the treatment and control effort gives the most cost effective combinations and at the same time the highest Robin [17] worked on cost effectiveness of a systematic one- off Chlamydia trachomatic (CT) screening program including partner treatment for Dutch young adults. Data on infection prevalence, participation rates and sexual behavior were obtained from a large pilot. Study conducted in the Netherland. Opposite to almost all previous economic evaluation of (CT) Screening, they developed a dynamic susceptible-infected susceptible (SIS) model to estimate the impact of the screening programme on the incidence and prevalence of (CT) in the population SIS model are widely used in epidemiology of infected disease for modeling. The transmission dynamic overtime subsequently-a predictive decision model was used to calculate the complication averted by the screening programme. Cost effectiveness was expressed as the net cost per major outcome averted (ANOA) and use estimated in the baseline analysis and in sensitivity analysis. [2, 4] developed a deterministic epidemiological model of (S, E, I, R) to gain insight into the efficacy and compliance of condom on the dynamical spread of Gonorrhea disease. Positivity solution was analyzed for mathematical and epidemiological posedness of the model and Local and global stability of the model were explored for disease-free and endemic equilibria. Sensitivity analysis is performed on basic reproduction number to check the importance of each parameter on the transmission of gonorrhea disease. Numerical simulation was analyzed by MAPLE 18 software using embedded Runge-Kutta method of order (4) which shows the effect of condom on the prevention/control of Gonorrhea disease.

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2. Model Formulation

A nonlinear mathematical model is formulated and analyzed to study the effect of condom efficacy, detection of undetected individual, effective contact rate, progression rate and other epidemiological features on the dynamical spread of Chlamydia infection. Considered six (6) compartment deterministic mathematical model using $S(t), E(t), I_{us}(t), I_A(t), I_{ds}(t), R(t)$ to have better understanding of efficacy of condom on Chlamydia disease. So that,

$$N(t) = S(t) + E(t) + I_{us}(t) + I_A(t) + I_{ds}(t) + R(t) \quad (1)$$

The Susceptible population is a member of a population who is at risk of becoming infected by a disease. The population of susceptible individual increase by the recruitment of sexually active or by birth of individual as a rate (π), The population decrease by natural death μ, also by infection following a contact with infected individual who did not use condom at rate β. The susceptible population later increased by recovered individual after the wanes treatment at rate ω Effective constant with infection individual at a rate λ

given by $\lambda = \beta \dfrac{(\eta_1 I_{us} + \eta_2 I_{ds} + \eta_3 I_A)}{N}$

Where β represent contact capable of leading to Chlamydia infection, $\eta_1\ \eta_2\ \eta_3$ are modification parameters that compare the transmissibility of the disease. Then the rate of change of susceptible population is given by

$$\frac{dS}{dt} = \pi - (1-\theta)\,\lambda\,\mathrm{S(t)} - \mu\mathrm{S(t)} + \omega\mathrm{R(t)} \quad (2)$$

Exposed (E): Exposed individual is a member of a population generated through infection of susceptible that infected but not yet infectious, Exposed individual increase through the infection of susceptible by the fraction $(1-\varepsilon)$ and are assumed to show no disease symptoms initially. The population of exposed individual is decreased by progression rate at (τ_2) of exposed individual to active infected undetected symptomatic (I_{us}) at the rate (γ) and also diminished by the natural death at a rate μ.

Finally increase through, the infection of susceptible and are assumed to slow no disease symptoms initially. Exposed individual is a member of a population who is infected but not infections at rate (γ) that moves from treated class to Exposed compartment

Thus

$$\frac{dE}{dt} = (1-\varepsilon)\lambda\mathrm{S(t)} - \theta\lambda S(t) - (1+\gamma)\tau_2 E(t) - \mu\mathrm{E(t)} \quad (3)$$

The population of undetected infected symptomatic individual is a member of population that develop symptom but cannot recognize the symptoms, It increased by remaining fraction (ε) and decreased by detection rate of infected

detected symptomatic (ρ). This population is also decreased by natural death rate (μ) and disease induced death (at a rate δ). Hence

$$\frac{dI_{us}}{dt} = \varepsilon\lambda \mathrm{S(t)} - \rho \mathrm{I_{us}(t)} - (\mu + \delta)\mathrm{I}_{us} \tag{4}$$

The population of asymptomatic individuals is member of population that not show symptoms of chlamydia at all, They increased by the infection progression at the rate (γ), which cause development of symptoms by exposed individual at the rate γ becoming symptomatic. This population is decreased by natural death rate (μ) and disease induced death (at a rate δ). Hence

$$\frac{dI_A}{dt} = \gamma\tau_2 \mathrm{E(t)} + \psi\tau_1 I_{ds}(t) - (\delta + \mu)I_A(t) \tag{5}$$

The population of Detected infected symptomatic chlamydia individual is a member of population who is infected and capable of transmitting the disease. The population of detected infected asymptomatic individual increase by the fraction of infection becoming symptomatic at rate (τ_2) is the endogenous reactivation rate and the detection of undetected individual at the rate ψ . The population later decreased by treatment rate (δ for detected individual and finally reduced by the natural death rate, induced mortality death rate at μ . Hence

$$\frac{dI_d}{dt} = \tau_2 E(t) + \rho \mathrm{I_{us}}(t) - (1+\psi)\tau_1 I_{ds} - (\mu + \delta)I_{ds}(t) \tag{6}$$

Recovered individual is a member a member of population who recovered from the disease. The population of Chlamydia recovered individual is increase by the treatment of detected individual at rate (τ_1) successfully recovered individual eventually more to the exposed class. This population later decrease by the natural death and individual that loss immunity since there is no permanent immunity to Chlamydia at rate (μ) and (ω) respectively.

$$\text{Hence } \frac{dR}{dt} = \tau_1 I_{ds}(t) - (\mu + \omega)R(t) \tag{7}$$

Putting all these assumptions together to obtain the below equation

$$\left.\begin{aligned}
\frac{dS}{dt} &= \pi - (1-\theta)\lambda S(t) - \mu S(t) + wR(t) \\
\frac{dE}{dt} &= (1-\varepsilon)\lambda S(t) - \theta\lambda S(t) - (1+\gamma)\tau_2 E(t) - \mu E(t) \\
\frac{dI_{us}}{dt} &= \varepsilon\lambda S(t) - (\mu+\delta+\rho) I_{us}(t) \\
\frac{dI_A}{dt} &= \gamma\tau_2 E(t) + \psi\tau_1 I_{sd} - (\mu+\delta) I_A(t) \\
\frac{dI_{ds}}{dt} &= \tau_2 E(t) + \rho I_{us}(t) - (1+\psi)\tau_1 I_{ds}(t) - (\mu+\rho) I_{ds} \\
\frac{dR}{dt} &= \tau_1 I_{ds}(t) - (\mu+w) R(t)
\end{aligned}\right\} \quad (8)$$

$$\lambda = \beta \frac{(\eta_1 I_{us} + \eta_2 I_{ds} + \eta_3 I_A)}{N} \quad (9)$$

Table 1: Description of Variables and Parameters

Parameters	Description
π	Recruitment rate into population
μ	Natural death rate
S	Susceptible individuals with Chlamydia
E	Exposed Chlamydia individual
I_{us}	Infected undetected symptomatic individual
I_A	Asymptomatic individual
I_{ds}	Infected detected symptomatic individual
R	Recovered individual

θ	Condom efficacy
ε	A fraction of Susceptible individuals in contact with individual capable of transmitting the disease, further has that a fraction of them show symptom but cannot recognize the disease while the remaining fraction does not show the symptom
τ_2	Progression rate of exposed individual
δ	Disease induced death rate for asymptomatic
τ_1	Treatment rate
ω	Loss of immunity of recovered individual
β	Contact rate
η_1, η_2	Modification parameter
ψ	Treated infected detected individual who are asymptomatic
γ	Fraction of progression exposed individual who are asymptomatic
ρ	Detection rate of infected undetected symptomatic individual
N	Total population
λ	Force of infection

2.1 Positivity of Solution

Theorem 1: the closed set $D = \{(S, E, I_{us}, I_A, I_{ds}, R) \in \Re_+^6 : N \leq \frac{\pi}{\mu}\}$ is positively invariant and attracting with respect to the model (8) coupled with equation (10) above .

Proof:

Consider the biologically –feasible region D, the rate of change of the total population is obtained by adding all the equations in equation (8) which give:

$$\frac{dN}{dt} = \pi - \mu s - \mu E - \mu I_{us} - \mu I_A - \mu I_{ds} - \mu R \quad (10)$$

Where $N = S+E+I_{us}+I_A+I_{ds}+R$

$$\frac{dN}{dt} = \frac{dS}{dt} + \frac{dE}{dt} + \frac{dI_{us}}{dt} + \frac{dI_A}{dt} + \frac{dI_{ds}}{dt} + \frac{dR}{dt} \quad (11)$$

$$\frac{dN}{dt} = \pi - \mu N - \delta I_{us} - \delta I_{ud} - \delta I_A$$

In the absent of infection, equation (11) implies

$$\frac{dN}{dt} = \pi - \mu N \quad (12)$$

$$\frac{dN}{dt} + \mu N = \pi \quad (13)$$

Using the integrating factor I.F= $e^{\int \mu dt}$

I.F= $e^{\int \mu dt} = e^{\mu t}$ (14)

Multiply both sides of equation (13) by equation (14), it implies:

$$e^{\mu t}\left\{\frac{dN}{dt}\right\} + \mu N(t)e^{\mu t} = \pi e^{\mu t} \quad (15)$$

$$= \left\{\frac{d}{dt}(Ne^{\mu t})\right\} = \pi e^{\mu t}$$

Integrate both sides with respect to *t*

$$Ne^{\mu t} = \frac{\pi}{\mu} + e^{\mu t} + K \quad \text{(where k is a constant of integration)} \quad (16)$$

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Multiply both sides of equation (16) by $e^{\mu t}$

It becomes:

$$N(t) = N(0)e^{-\mu t} + \frac{\pi}{\mu}(1 - e^{-\mu t}) \tag{17}$$

$$N \to \frac{\pi}{\mu} \text{ as } t \to \infty$$

Therefore $\frac{dN}{dt} < 0$ wherever the total population $N > \frac{\pi}{\mu}$. Hence, for all $t>0$, all the solutions of the model with the initial conditions in region D will remain in the region D (where the model can be considered as being epidemiologically and mathematically well posed). Thus the biologically feasible region D is positively-invariant and attracting.

2.2 Disease free equilibrium

At critical points, disease equilibrium is obtained as;

$$\varepsilon_0 = \left\{\frac{\pi}{\mu}, 0, 0, 0, 0, 0\right\}$$

2.3 Existence of Endemic Equilibrium

Let $\varepsilon_0^* = (S^{**}, E^{**}, I_{us}^{**}, I_A^{**}, I_{ds}^{**}, R^{**})$ are the endemic equilibrium points.

From equation (8) make $E^{**}, I_{us}^{**}, I_A^{**}, I_{ds}^{**}, R^{**}$ as the subject to obtain the following

$$E^{**}(t) = \frac{(1-\varepsilon)-\theta)\lambda^{**}S^{**}}{k_1},\ Ius^{**}(t) = \frac{\varepsilon\lambda^{**}S^{**}}{k_2},\ I_A^{**}(t) = \frac{\gamma\tau_2 E^{**} + \psi\tau_1 I_{ds}^{**}}{k_3}$$

$$, I_{ds}^{**} = \frac{\tau_2 E^{**} + S^{**} I_{us}^{**}}{k_4}, R^{**} = \frac{\tau_1 I_{ds}^{**}}{k_5} \tag{18}$$

Re-write equation (18) in term of λ^{*} S^{**} after the manipulation obtain the following as;

$$E^{**} = P_1\lambda^{**}S^{**} \text{ where } P_1 = \frac{(1-\varepsilon)+\theta}{k_1}$$

$$I_u^{**} = P_2\lambda^{**}S^{**}, \text{ where } P_2 = \frac{\tau_2 E^{**} + \rho I_u^{**}}{k_4} \quad (19)$$

$$I_d^{**} = P_3\lambda^{**}S^{**}, \text{ where } P_3 = \frac{\gamma I_{ds}^{**}}{k_5}$$

$$R^{**} = P_5\lambda^{**}S^{**}, \text{ where } R^{**} = \frac{\gamma I_{ds}^{**}}{k_5}$$

Now, substitute equation (19) into (9), upon simplification the following was obtained

$$\lambda^{**} = \frac{R_0 - 1}{P_6} > 0, \text{ whenever } R_0 > 1.$$

where

$$K_1 = (\tau_2(1+\gamma)+\mu), \quad K_2 = (\rho+\delta+\mu), \quad K_3 = (\mu+\delta), K_4 = (\tau_1(1-\psi)+\delta+\mu), \quad K_5 = (\mu+\omega)$$

The component of the unique endemic equilibrium (R_0) can be obtained by substituting the unique value of λ^{**} into expression in (18) thus, the result is established.

2.4 Basic reproduction number

The basic reproduction number R_0 of chlamydia disease model (8) is calculated by using the next generation matrix [5]. This is obtained after taking partial derivative F and V given as;

$$F := \begin{bmatrix} 0 & \frac{(1-\xi-\theta)\beta\eta_1\pi}{\mu} & \frac{(1-\xi-\theta)\beta\eta_3\pi}{\mu} & \frac{(1-\xi-\theta)\beta\eta_2\pi}{\mu} & 0 \\ 0 & \frac{\xi\beta\eta_1\pi}{\mu} & \frac{\xi\beta\eta_3\pi}{\mu} & \frac{\xi\beta\eta_2\pi}{\mu} & 0 \\ 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \end{bmatrix}; \quad V = \begin{bmatrix} K_1 & 0 & 0 & 0 & 0 \\ 0 & K_2 & 0 & 0 & 0 \\ -\gamma\tau_2 & 0 & K_3 & -\psi\tau_1 & 0 \\ -\tau_2 & -\rho & 0 & K_4 & 0 \\ 0 & 0 & 0 & -\tau_1 & K_5 \end{bmatrix}$$

where

$K_1 = \tau_2(1+\gamma)+\mu,\ K_2 = (\rho+\delta+\mu)\phi,\ K_3 = (\mu+\delta), K_4 = (\mu+\delta(1+\psi)\tau_1)$, $K_5 = (\mu+\omega)$

Therefore, the chlamydia disease reproduction number is

$$R_0 = \frac{1}{\mu K_2 K_4 K_3 K_1}(\pi\beta(\gamma\theta K_2 K_4\eta_3\tau_2 + \gamma\xi K_2 K_4\eta_3\tau_2 - \psi\rho\xi K_1\eta_3\tau_1 + \psi\theta K_2\eta_3\tau_1\tau_2 - \gamma K_2 K_4\eta_3\tau_2$$
$$-\psi K_2\eta_3\tau_1\tau_2 - \rho\xi K_1 K_3\eta_2 + \theta K_2 K_3\eta_2\tau_2 - \xi K_1 K_3 K_4\eta_1 + \xi K_2 K_3\eta_2\tau_2 - K_2 K_3\eta_2\tau_2)$$

2.5 Global stability of endemic equilibrium

Theorem1: for *Ro>1* then equation` (8) is globally systemically stable if S =S*, E =E*, $I_u = I_{us}^*, I_A = I_A^*, I_{ds} = I_{ds}^*$, $R = R^*$ and $M < N$ and unstable $R \le 1$

Proof:

Using the constructed Iyapunov function by (Cai and Li, 2000) the global stability is done using Lyapunov function as follow

$$V = (S^*, E^*, I_{us}^*, I_A^* I_{ds}^* R^*) = (S - S^* - S^* Log\frac{S}{S^*}) + \left(E - E^* - E^* Log\frac{E}{E^*}\right) + \left(I_{us} - I_{us}^* - I_{us}^* Log\frac{I_{us}}{I^*_{us}}\right) +$$
$$+\left(I_A - I_A^* - I_A^* Log\frac{I_A}{I^*_A}\right) + \left(I_{ds} - I_{ds}^* - I_{ds}^* Log\frac{I_{ds}}{I^*_{ds}}\right) + \left(R - R^* - R^* Log\frac{R}{R^*}\right)$$

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By direct calculating, the derivation of lyapunov the solution of equation (1) – (6) we have

$$\frac{dv}{dt}=\frac{ds}{dt}-\left(\frac{S^*}{S}\right)\frac{ds}{dt}+\frac{dE}{dt}-\left(\frac{E^*}{E}\right)\frac{dE}{dt}+\frac{dI_{us}}{dt}-\left(\frac{I_{us}^*}{I_{us}}\right)\frac{dI_{us}}{dt}+\frac{dI_A}{dt}-\left(\frac{I_A^*}{I_A}\right)\frac{dI_A}{dt}+\frac{dI_{ds}}{dt}-\left(\frac{I_{ds}^*}{I_{ds}}\right)\frac{dI_{ds}}{dt}+\frac{dR}{dt}-\left(\frac{R^*}{R}\right)\frac{dR}{dt}$$

$$\frac{dv}{dt}=\left((\pi-(1-\theta))\lambda S-\mu S+wR\right)-\left(\frac{S^*}{S}\right)\left((\pi-(1-\theta))\lambda S-\mu S+wR\right)+\left(1-\varepsilon)-\theta\right)\lambda S-\left(\mu+(1+\gamma)\tau_2\right)E\right)$$

$$-\left(\frac{E^*}{E}\right)\left(1-\varepsilon)-\theta\right)\lambda S-\left(\mu+(1+\gamma)\tau_2\right)E\right)+\varepsilon\lambda S-\left(\mu+\delta+\rho\right)I_{us}+\left(\frac{I_{us}^*}{I_{us}}\right)\varepsilon\lambda S-\left(\mu+\delta+\rho\right)I_{us}+\gamma\tau_2E(t)+\psi\tau_1I_{ds}-(\mu+\delta)I_A$$

$$-\left(\frac{I_A^*}{I_A}\right)\gamma\tau_2E(t)+\psi\tau_1I_{ds}-(\mu+\delta)I_A+\left(\frac{I_{ds}^*}{I_{ds}}\right)\left(\tau_2E+\rho I_u-\left(\mu+\delta(1+\psi)\tau_1\right)I_{dS}\right)+\tau_1I_{ds}-\left(\mu+w\right)-\left(\frac{R^*}{R}\right)\left(\tau_1I_{ds}-\left(\mu+w\right)\right)$$

It implies that

$$\frac{dv}{dt}=\left(\pi-(1-\theta)\right)\lambda S-\mu S+wR-\pi\left(\frac{S^*}{S}\right)+(1-\theta)\lambda S^*+\mu S*-wR\frac{S^*}{S}+\left(1-\varepsilon)-\theta\right)\lambda\left(S\right)-K_1E$$

$$-\left(\frac{E^*}{E}\right)\left(1-\varepsilon)-\theta\right)\lambda\left(S\right)+K_1E*+\varepsilon\lambda S\left(\frac{I_{us}^*}{I_{us}}\right)+K_2I*_{us}+\gamma\tau_2E+\psi\tau_1I_{ds}-K_3I_A-\gamma\tau_2\varepsilon\lambda\frac{I_A^*}{I_A}-\psi\tau_1EI_{ds}(\tau+\mu)(I_A)+$$

$$K_3I_A^*+\tau_2E+\delta I_{us}-K_4I_{ds}-\tau_2E\left(\frac{I_{ds}^*}{I_{ds}}\right)-\delta\frac{I_{ds}^*}{I_{ds}}+K_4I_{ds}^*+\tau_1\left(I_{ds}\right)-K_5R-\tau_1I_{ds}\left(\frac{R^*}{R}\right)+K_5R^*$$

Collect like terms

$$\frac{dv}{dt}=\pi+wR+\left((1-\theta)\right)\lambda S*+\mu S*+K_1E*+\varepsilon\lambda S\left(\frac{I_{us}^*}{I_{us}}\right)+K_2I*_{us}+\gamma\tau_2E+\psi\tau_1I_{ds}+K_3I_A^*+\tau_2E+\delta I_{us}+K_4I_{ds}^*+\tau_1\left(I_{ds}\right)+K_5R^*$$

$$-(1-\theta)\lambda S-\mu S-K_1E-\left(\frac{E^*}{E}\right)\left(1-\varepsilon)-\theta\right)\lambda\left(S\right)-K_3I_A-\gamma\tau_2\varepsilon\lambda\frac{I_A^*}{I_A}-\psi\tau_1EI_{ds}(\tau+\mu)(I_A)-K_4I_{ds}-\tau_2E\left(\frac{I_{ds}^*}{I_{ds}}\right)-\delta\frac{I_{ds}^*}{I_{ds}}$$

$$-K_5R-\tau_1I_{ds}\left(\frac{R^*}{R}\right)$$

Re-arrange the positive and negative terms

$$\frac{dv}{dt} = M - N$$

$$M = \pi + wR + ((1-\theta))\lambda S^* + \mu S^* + K_1 E^* + \varepsilon\lambda S\left(\frac{I_{us}^*}{I_{us}}\right) + K_2 I^*_{us} + \gamma\tau_2 E + \psi\tau_1 I_{ds} + K_3 I_A^* + \tau_2 E + \delta I_{us} + K_4 I_{ds}^* + \tau_1(I_{ds}) + K_5 R^*$$

$$N = (1-\theta)\lambda S - \mu S - K_1 E - \left(\frac{E^*}{E}\right)(1-\varepsilon)-\theta))\lambda(S) - K_3 I_A - \gamma\tau_2\varepsilon\lambda\frac{I_A^*}{I_A} - \psi\tau_1 E I_{ds}(\tau+\mu)(I_A) - K_4 I_{ds} - \tau_2 E\left(\frac{I_{ds}^*}{I_{ds}}\right) - \delta\frac{I_{ds}^*}{I_{ds}}$$

$$- K_5 R - \tau_1 I_{ds}\left(\frac{R^*}{R}\right)$$

Hence, if M < N, note that $\frac{dv}{dt} = 0$ if and only if $S = S^*,\ E = E^*, I_{us} = I_{us}^*, I_A = I_A^*, I_{ds} = I_{ds}^*, R = R^*$, therefore the largest compact in variant set $\left\{\left(S^*, E^*, I_{us}^*, I_A^*, I_{ds}^*, R^*\right) \in \Gamma, \frac{dv}{dt} = 0\right\}$ is the singleton $\{\varepsilon^*\}$ where ε^* is the endemic equilibrium. Hence by the Lasalle's invariant principle, it implies that ε^* is globally asymptotically stable in Γ if M <

3. Numerical simulation of result

Numerical Stimulation was carried out by MAPLE 18 software using Range-kutta method of order four with the set of parameter values given in the Table 2 and the following Figures below are obtained:

$S(0) = 500,\ E(0) = 300,\ I_{us}(0) = 200,\ I_A(0) = 150,\ I_{Ds}(0) = 100,\ R(0) = 50$

Table 2:Parameters value used for the numerical simulation

Symbol	Value	Source
π	3.12	[8]
μ	0.02	[17]
θ	3.99	Assumed
γ	0.10	[4]
	1.00	[8]
η_1	0.10	Assumed
τ_1	0.7	Assumed
ε	0.01	[10]
β	0.22	Assumed
	0.30	Assumed
ρ	0.02	[8]
ω	0.43	Hamilton
ψ	0.7	[4]
η_2	0.01	[6]
τ_2		

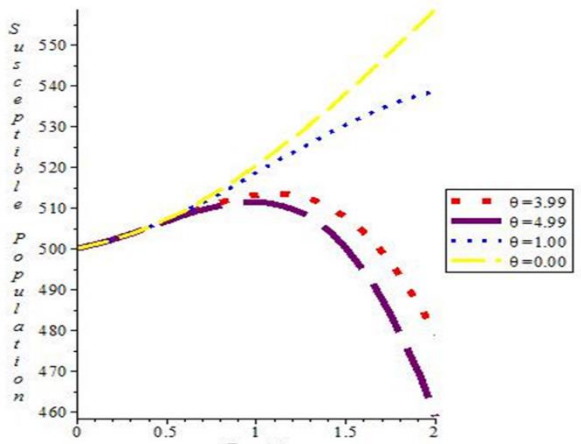

Figure:1 Graph of susceptible individual for various values of condom efficacy

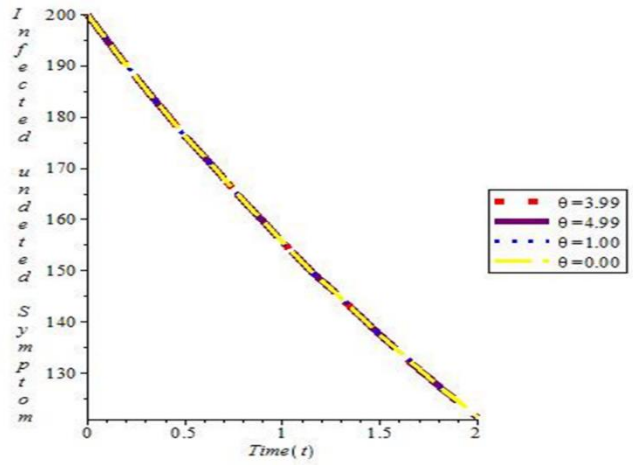

Figure 2: Graph of infected undetected symptomatic individual for various values of condom efficacy

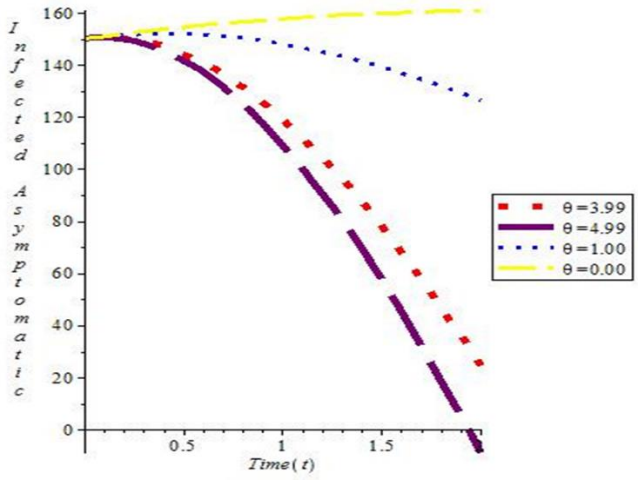

Figure3: Graph of infected asymptomatic individual for various values of condom efficacy

4. Discussion of Results

In this study, six (6) compartmental epidemiological models of (S, E, I_{us}, I_A, I_{ds}, R) are presented to gain insight into the efficacy of condom on the dynamical spread of chlamydia disease. Positivity of solution shows that, the model presented is mathematically and epidemiologically well posed. Global stability of the model show that the threshold quantity "R_o" is less than unity and otherwise endemic when it is greater than unity. In Figures 1, 2 and 3 of numerical simulation graphs showed that, increasing the rate of efficacy of condom reduces the susceptible, infected undetected symptomatic and infected asymptomatic individuals. In conclusion, the use of condom with total compliance can reduce the spread of chlamydia disease increment in the values of condom efficacy and also reduced the basic reproduction number "Ro" since the spread of disease is dependent on the values of "Ro". Therefore, efforts that target the use of condom as a control measure should be encouraged.

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